

Appl. No. : **09/937,365**
Filed : **January 18, 2002**

REMARKS

Claims 1, 25 and 37 have been amended to delete the term “prevent” or to change the term “prevent” to “treat” without prejudice, in accordance with the Examiner’s requirement. Claim 31 has been amended to delete the term “with high serum IgE level”. The amendments neither raise the issue of new issue nor the addition of new matter to the application. Applicant respectfully requests entry of the amendments and reconsideration of the application in view of the amendments and the following remarks.

Rejection of Claims 1, 10-22, 25-30 and 37-42 Under 35 U.S.C. § 112

Claims 1, 10-22, 25-30 and 37-42 have been rejected under 35 U.S.C. § 112, first paragraph, with regard to enablement for “prevention of type I allergies”.

Since Claims 10-15 are dependent ultimately on Claim 24 which recites “treatment” (not prevention) of pollinosis, the rejection of these claims is an error. Claims 1, 25 and 37 have been amended to delete the term “prevent” or to change the term “prevent” to “treat” without prejudice, in accordance with the Examiner’s requirement. The term “treatment” should be interpreted to include “prevention” in a broad sense. The remaining claims are dependent ultimately on either one of Claims 1, 25 and 37. Thus, it is respectfully requested that the rejection be withdrawn.

Rejection of Claims 25-30 and 37-42 Under 35 U.S.C. § 112

Claims 25-30 and 37-42 have been rejected under 35 U.S.C. 112, second paragraph, with regard to the indefinite terms.

As described above, Claims 25 and 37 have been amended to change the term “prevent” to “treat”. The remaining claims are dependent ultimately on either Claim 25 or 37. Thus, it is respectfully requested that the rejection be withdrawn.

Rejection of Claims 1, 10-22 and 24-42 Under 35 U.S.C. § 103

Claims 1, 10-22 and 24-42 have been rejected under 35 U.S.C. § 103(a) as being unpatentable over H. Fukumoto et al. in combination with Sawruk (US 5,478,579). Applicant respectfully traverses this rejection. Claims 1, 24, 25, 31 and 37 are independent and the

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remaining claims are dependent ultimately on either one of Claims 1, 24, 25, 31 and 37. The claims could not be obvious over the combination of H. Fukumoto et al. and Sawruk as explained below.

Claims 1, 24 and 31

MPEP § 706.02(j) states: "To establish a *prima facie* case of obviousness, three basic criteria must be met. First, there must be some suggestion or motivation, either in the references themselves or in the knowledge generally available to one of ordinary skill in the art, to modify the reference or to combine reference teachings. Second, there must be a reasonable expectation of success. Finally, the prior art reference (or references when combined) must teach or suggest all the claim limitations. The teaching or suggestion to make the claimed combination and the reasonable expectation of success must both be found in the prior art and not based on applicant's disclosure. *In re Vaeck*, 947 F.2d 488, 20 USPQ2d 1438 (Fed. Cir. 1991)."

Here, the Examiner asserts:

- (1) "Fukumoto teaches kaempferol-3-glucoside is known as an anti-anaphylactic agent."
- (2) "Further, the compounds taken from white petals of *impatiens balsamina* L. inhibit IgE-mediated anaphylaxis."
- (3) "The compounds extracted were used to treat allergic reactions on skin."
- (4) "Fukumoto further teaches that astragalin is known to inhibit the release of IgE-promoted histamines."

However, the Examiner fails to show that the reference teaches or suggest "atopic dermatitis" or "pollinosis". Atopic dermatitis or pollinosis is not the same as anaphylaxis or mere allergic reactions on skin. No reference teaches that atopic dermatitis or pollinosis is equivalent to anaphylaxis or mere allergic reactions on skin. Inhibiting "IgE-mediated anaphylaxis" and "the release of IgE-promoted histamines" is too general to determine that atopic dermatitis or pollinosis is equivalent to anaphylaxis. Each symptom involves specific IgE. Further, the exact cause of atopic dermatitis is not known (see page 4 of the attached copy of eMedicine-Dermatitis, Atopic: Article by Anthony J Ghidorzi, Jr., Do, www.emedicine.com/emerg/topic130.htm). Further, antihistamines or other drugs are not greatly effective (see page 8). Also, antihistamine agents do not have a significant influence on nasal congestion caused by pollinosis (see page 1 of the attached copy of Principles of diagnostics and

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therapy of pollinosis in the pre-school children – J. Hofman, www.radoslawspiewak.net/pp_082.htm). Furthermore, pollinosis involves humoral immunity (IgE, allergen specific IgE and IgG4, anti-IgE), cellular immunity (activity and accumulation of effector cells and release of allegro-inflammatory mediators), and generation of the late allergic reaction phase with participation of eosinophils, neutrophils, basophils and T cells, in particular Th2 (see page 1 of the attached copy of The specific immunotherapy in pollinosis – S. Chyrek - Borrowaska (1995), www.radoslawspiewaknet/pp_093.htm). In view of the foregoing, one of ordinary skill in the art could not reasonably conclude that Fukumoto (or Sawruk) teaches or suggests all the claim limitations, especially, atopic dermatitis or pollinosis.

Further, the Examiner oversimplifies Fukumoto's disclosure: The Examiner asserts "Further, the compounds taken from white petals of *impatiens balsamina* L. inhibit IgE-mediated anaphylaxis (See Introduction)." However, Fukumoto's introduction states "IB [a 35% ethanol extract of petals] significantly inhibited the fatal shock of hen egg-white lysozyme (HEL) specific IgE-mediated anaphylaxis in ddY mice." (Emphasis added.) Fukumoto describes very specific symptoms, which could not generally apply to atopic dermatitis or pollinosis. The Examiner fails to show that the fatal shock of hen egg-white lysozyme (HEL) specific IgE-mediated anaphylaxis in ddY mice is applicable to atopic dermatitis or pollinosis.

Additionally, the Examiner asserts "Fukumoto further teaches that astragalin is known to inhibit the release of IgE-promoted histamines (Page 205, second column)." However, Page 205, second column reads "kaempferol-3-glucoside (7) have been reported to inhibit the release of IgE-promoted histamine from mast cells." (Emphasis added.) Here, also, Fukumoto describes a very specific phenomenon, which could not generally apply to atopic dermatitis or pollinosis. The Examiner fails to show that the release of IgE-promoted histamine from mast cells is applicable to atopic dermatitis or pollinosis. The Examiner is required to show a reference teaching the above or the knowledge generally available to one of ordinary skill in the art pursuant to MPEP § 706.02(j); otherwise, the Examiner could not maintain this rejection.

No reference teaches or suggests that kaempferol-3-glucoside (astragalin) is effective in treating atopic dermatitis or pollinosis. As described above, antihistamine agents do not have a significant influence on nasal congestion caused by pollinosis (page 1 of Principles of diagnostics and therapy of pollinosis in the pre-school children – J. Hofman). In the instant specification,

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Example 5 (page 29, Table 1) shows that astragalin significantly worked on nasal congestion as well. Further, as described above, antihistamines or other drugs are not greatly effective for atopic dermatitis (page 8 of eMedicine-Dermatitis, Atopic: Article by Anthony J Ghidorzi, Jr., Do). However, in the instant specification, Example 3 (page 24-26) shows astragalin significantly worked on atopic symptoms. There could not be a reasonable expectation of success for one of ordinary skill in the art.

Lastly, Sawruk is irrelevant to the above discussion.

In view of the foregoing, a combination of H. Fukumoto et al. and Sawruk could not render Claims 1, 24 or 31 obvious.

Claims 25 and 37

Claims 25 and 37 include recitations similar to or the same as those in Claims 1, 24, and 31. Thus, at least for the reason above, a combination of H. Fukumoto et al. and Sawruk also could not lead to the invention of Claims 25 or 37.

In addition, none of the cited references teaches or even suggests (i) administering astragalin prior to the season for treating pollinosis and (ii) continuing administering astragalin to treat pollinosis until the season is over, as recited in Claim 25. None of the cited references teaches or even suggests administering astragalin before showing symptoms thereof for treating atopic dermatitis as recited in Claim 37. The Examiner fails to show that all of the above recitations are taught or suggested by the prior art references and that one of ordinary skill in the art would have been motivated to employ the above recitations. Therefore, Claims 25 and 37 could not be obvious over the prior art references.

The remaining claims which depend on either one of Claims 1, 24, 25, 31, or 37 include the recitations discussed above.

In view of the foregoing, Claims 1, 24, 25, 31 and 37 and the claims dependent thereon could not be obvious over the references. Applicant respectfully requests withdrawal of this rejection.

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CONCLUSION

In light of the Applicant's amendments to the claims and the foregoing Remarks, it is respectfully submitted that the present application is in condition for allowance. Should the Examiner have any remaining concerns which might prevent the prompt allowance of the application, the Examiner is respectfully invited to contact the undersigned at the telephone number appearing below.

Please charge any additional fees, including any fees for additional extension of time, or credit overpayment to Deposit Account No. 11-1410.

Respectfully submitted,

KNOBBE, MARTENS, OLSON & BEAR, LLP

Dated: October 20, 2004

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Dermatitis, Atopic

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Last Updated: October 5, 2004

Synonyms and related keywords: eczema, ichthyosis vulgaris, keratosis pilaris, hand and foot dermatitis, keratoconus

AUTHOR INFORMATION

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INTRODUCTION

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Background: Atopic dermatitis (ie, eczema) is a chronic pruritic skin condition usually beginning in infancy.

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[Dermatitis, Contact](#)[Dermatitis, Exfoliative](#)[Scabies](#)

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Patient Education

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Pathophysiology: Precise etiology is unknown, but current theories center on a disordered immune response, especially an imbalance of cytokines. The disease also appears to have a hereditary component; family history is positive for atopy (ie, asthma, allergic rhinitis, atopic dermatitis) in two thirds of patients.

Eczema Treatment

Frequency:

- **In the US:** Prevalence of atopic dermatitis is 12%.

Mortality/Morbidity: This disease usually is not life threatening.

Sex: No predilection for occurrence exists; however, females have a worse prognosis.

Age: Eczema typically manifests in infants aged 1-6 months. Approximately 60% of patients experience their first outbreak by age 1 year and 90% by age 5 years. Onset of atopic dermatitis in adolescence or later is uncommon and should prompt consideration of another diagnosis. Disease manifestations vary with age.

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History:

- The emergency physician often is the first to diagnose eczema. The most common presentation is that of infants, usually younger than 6 months, brought in by their parents for a persistent rash. Before coming to the ED, the parents may have tried a number of over-the-counter and home remedies. Parents usually report that the rash has waxed and waned for months and a history of dry skin since birth.
- The predominant symptom is intense pruritus. Atopic dermatitis typically is not associated with fever or other constitutional symptoms. Dermatologic manifestations of eczema usually occur on the cheeks, diaper area, hands, and extensor surfaces in children. The antecubital and popliteal fossae often are involved in adults. Older children and adults presenting to the ED usually know of their diagnosis but present because of a flare-up or complication.
- Distribution of lesions
 - Infants - Trunk, face, and extensor surfaces
 - Children - Antecubital and popliteal fossae

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- Adults - Face, neck, upper chest, and genital areas (In adults with limited distribution of lesions, a history of childhood eczema aids in diagnosis.)

Physical:

- Skin findings depend on the stage of disease.
 - Acute - Erosions with serous exudate or intensely itchy papules and vesicles on an erythematous base
 - Subacute - Characterized by scaling, excoriated papules, or plaques over erythematous skin
 - Chronic - Recognized by the presence of lichenification and pigmentary changes (increased or decreased) with excoriated papules and nodules.
- Lesions may become secondarily infected because of scratching.
- Infected lesions present with the characteristic yellow crusting observed with impetigo or the surrounding erythema characteristic of cellulitis.
- Distribution of lesions
 - Infants: Atopic dermatitis is characterized by symmetric lesions over cheeks, forehead, scalp, trunk, and the extensor surfaces. Lesions may extensively involve the flexural surfaces, sparing only the diaper area. Scalp involvement may be severe enough to cause alopecia.
 - Children: Atopic dermatitis is characterized by symmetric lesions on wrists, ankles, and flexor areas of the extremities. Rash usually is reported as better in warmer months and worse in winter and fall. Generalized eruptions also may occur in this age group.
 - Adults: Atopic dermatitis primarily involves the flexor areas of the arms, legs, and neck. Axillary, groin, and intergluteal involvement is uncommon and should raise suspicion of another diagnosis.
- Other manifestations
 - Ichthyosis vulgaris is observed in one third or more of patients with atopic dermatitis. Characterizing features are hyperlinear palms and soles and polygonal fishlike scales, especially on the lower legs.

- Keratosis pilaris is characterized by asymptomatic horny follicular papules on the extensor surfaces of the upper arms, buttocks, and anterior thighs.
- Hand and foot dermatitis may be the only manifestation in adults and adolescents. Fissuring of the palms, soles, and fingers often occurs.
- Keratoconus is observed in severe cases. A cone-shaped cornea (requiring corneal transplant) may develop in the second or third decade of life.
- Associated features
 - Facial erythema
 - Perioral pallor
 - Infraorbital fold (ie, Dennie-Morgan line)
 - Dry skin
 - Increased palmar linear markings
 - Pityriasis alba (hypopigmented asymptomatic areas on face and shoulders)
 - Pilaris

Causes:

- Exact etiology is unknown but several triggers have been identified. Anything that could dry the skin may exacerbate atopic dermatitis. Potential triggers include excessive bathing, hand washing, lip licking, sweating, or swimming.
- Contact with solvents, detergents, deodorants, cosmetics, and soaps can exacerbate the disease. Loose or poorly fitting clothing that constantly rubs the skin also can cause problems.
- Excessive or prolonged heat may trigger a flare-up. Examples include hot showers or baths, overdressing, use of electric blankets or heating pads, and exposure to high humidity.
- Risk factors
 - Skin infections
 - Emotional stress

- Irritating clothes and chemicals
- Excessively hot or cold climate
- Food allergy in children (controversial)
- Exposure to tobacco smoke

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Other Problems to be Considered:

Ataxia-telangiectasia syndrome
Histiocytosis X
Lichen simplex chronicus
Photosensitivity rashes
Psoriasis
Wiskott-Aldrich syndrome
Seborrheic dermatitis (particularly in infants)
Ichthyosis vulgaris

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Lab Studies:

- Laboratory tests are not helpful for diagnosing atopic dermatitis; however, they may exclude other disorders. Diagnosis is based on history and physical examination.
- Serum immunoglobulin E (IgE) levels usually are elevated.

Procedures:

- Biopsy: Pathologic findings include a thickened and hyperkeratoid epidermis with the dermis showing perivascular inflammation.

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Emergency Department Care:

- Prevention is the mainstay of treatment for atopic dermatitis. The emergency physician must properly diagnose, educate, and treat acute exacerbations or complications. If secondary infection is present, initiate antibiotic therapy along with measures to control inflammation.
- Educate patients and their parents about common triggers and the necessity of avoiding them. Explain that no cure exists, but exacerbations can be minimized with proper skin care. Instruct patients to take short baths or showers in lukewarm water and limit the use of soap on axilla, groin, and feet. Recommend bathing on alternate days during the winter. Liberal use of a good skin moisturizer cannot be overemphasized, especially in winter and immediately after bathing or swimming. Emphasize minimizing contact with cosmetics, deodorants, detergents, solvents, or other known triggers.
- Use lubricants to keep as much moisture in the skin as possible. The best lubricant is also the greasiest. Patients can choose which moisturizer works best for them. Infants and children tolerate petroleum jelly, but adults are usually unwilling to use such a thick, greasy preparation. Using a moisturizing soap is recommend.
- Treat acute attacks with a low-to-moderate potency topical corticosteroid. Generic over-the-counter hydrocortisone (1%) often is sufficient. Hydrocortisone 2.5% is available by prescription. Inform the patient about adverse effects of topical steroids (eg, atrophy, hypopigmentation, striae, telangiectasia, thinning of the skin). Medium-to-high potency topical steroids should not be used on the face or neck area because of these potential adverse effects.
- Methods of corticosteroid delivery
 - Ointments retain the most water in skin while delivering the medication with a minimum of potentially irritating additives or preservatives. Many patients find the greasy nature of ointments difficult to tolerate.
 - Lotions are much thinner but trap less water in the skin than ointments. They are particularly useful on hair-bearing areas.
- Patients with severe flare-ups or weeping lesions may benefit from a 7-day course of oral prednisone (40-60 mg/d for adults, 1 mg/kg/d for children). As the lesions begin to dry, apply a topical steroid.
- To control itching, many dermatologists place patients on an antihistamine, an adjunct to education, lubrication, and corticosteroids. The sedative effects may be more beneficial than its antipruritic properties. In eczema, because the pruritus is not histamine mediated, it does not respond well to histamine blockade. However, patients have a tendency to scratch in their sleep, and sedating them with an antihistamine at bedtime reduces scratching. Because scratching plays a major role in perpetuating

rash, reducing it can improve the rash.

- Additional general measures
 - Decrease stress, if possible
 - Avoid irritating agents (ie, wool, perfumes)
 - Minimize sweating
 - Allow sun exposure
 - Humidify the house

Consultations:

- Because eczema is likely to be a lifelong disease, refer patients to a dermatologist or their primary care physician for ongoing care.
- Dermatologic consultation is especially important for severe cases or those that are secondarily infected.

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Drug regimens for eczema should include topical steroids, possibly an antihistamine, and an antibiotic if needed.

Drug Category: *Topical steroids* -- For most ED patients, a low-to-medium potency steroid that is available over the counter is adequate.

Drug Name	Hydrocortisone 1% (Westcort, Dermacort, Cortaid) -- An example of a low-potency topical steroid available OTC. An adrenocorticosteroid derivative suitable for application to skin or external mucous membranes. Has mineralocorticoid and glucocorticoid effects resulting in anti-inflammatory activity.
Adult Dose	Apply bid to affected areas
Pediatric Dose	Apply as in adults
Contraindications	Documented hypersensitivity; viral, fungal, and bacterial skin infections
Interactions	None reported
Pregnancy	C - Safety for use during pregnancy has not been established.

Precautions	Prolonged use, applying over large surface areas, application of potent steroids, and occlusive dressings may increase systemic absorption of corticosteroids and may cause Cushing syndrome, reversible HPA axis suppression, hyperglycemia, and glycosuria
Drug Name	Triamcinolone 0.1% (Aristocort) -- A medium potency topical steroid. Treats inflammatory dermatosis responsive to steroids. Decreases inflammation by suppressing migration of polymorphonuclear leukocytes and reversing capillary permeability.
Adult Dose	Apply bid to affected area
Pediatric Dose	Apply as in adults
Contraindications	Documented hypersensitivity; fungal, viral, and bacterial skin infections
Interactions	None reported
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Multiple complications (eg, severe infections, hyperglycemia, edema, osteonecrosis, myopathy, peptic ulcer disease, hypokalemia, osteoporosis, euphoria, psychosis, myasthenia gravis, growth suppression) may occur; abrupt discontinuation of glucocorticoids may cause adrenal crisis

Drug Category: *Antihistamines* -- Used more for sedating effect than antipruritic effect. Patients in deep sleep usually do not scratch.

Drug Name	Hydroxyzine (Atarax, Vistaril) -- Antagonizes H1-receptors in the periphery. Also may suppress histamine activity in the subcortical region of the CNS. A sedating antihistamine.
Adult Dose	25-50 mg PO q4-6h prn
Pediatric Dose	0.5 mg/kg PO q4-6h prn
Contraindications	Documented hypersensitivity
Interactions	CNS depression may increase with alcohol or other CNS depressants
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Associated with clinical exacerbations of porphyria (may not be safe for porphyric patients); ECG abnormalities (alterations in T-waves) may occur; may cause drowsiness

FOLLOW-UP

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Complications:

- Because of frequent scratching and fissuring of the skin, secondary infection is not uncommon. Suspect infection in persons with fever, surrounding erythema, or yellow crusting of the lesions. In severe exacerbations, it may be difficult to identify signs of secondary infection; empirical treatment for infection may be warranted.
- Eczema herpeticum: Herpes simplex may produce widespread lesions in patients with eczema. Suspect this diagnosis if multiple grouped vesicles develop with associated fever.
- Exfoliative dermatitis: Eczema may be the underlying disorder in this diffuse warm erythematous dermatitis that affects the entire body. Hospital admission is usually required.
- Cataracts are more common in patients with atopic dermatitis.
- Atrophy or striae occur if fluorinated corticosteroids are used on the face or in skin folds.
- Systemic absorption of steroids may occur if large areas of skin are treated, particularly if high-potency medications and occlusion are combined.

Prognosis:

- With the above therapeutic strategy, most patients improve; however, they need to understand that no cure is available. By following prevention strategies, the incidence of exacerbations can be minimized.
- Chronic disease tends to fade with age. About 90% of patients have spontaneous resolution by puberty.
- Some adults may continue to have localized eczema (eg, chronic hand or foot dermatitis, eyelid dermatitis, lichen simplex chronicus).

Patient Education:

- Instruct patients about proper skin care. Patients should keep bathing to a minimum, keep skin well lubricated, use mild soaps, and avoid known triggers.
- The goal is control, not cure.
- For excellent patient education resources, visit eMedicine's [Skin, Hair, and Nails Center](#). Also, see eMedicine's patient education article [Eczema](#).

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Special Concerns:

- Given the chronic nature of this disease and patients' concerns about appearance, emotional support and psychological counseling may be helpful. Physicians need to be sensitive to the needs of patients and their families.

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R. Spiewak (Editor): "Pollens and Pollinosis: Current Problems"

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Pages 82-83

PRINCIPLES OF DIAGNOSTICS AND THERAPY OF POLLINOSIS IN THE PRE-SCHOOL CHILDREN

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The epidemiological investigations show that allergic rhinitis occurs in about 12-20% of population. Analysing these changes regarding all allergic symptoms during the season, patients with allergic rhinitis can constitute 40-60% of all treated, especially those over 7 years of age, in whom symptoms of allergic rhinitis prevail among systemic allergoses' manifestations. Not lesser diagnostic and therapeutical difficulties apply to the pre-school children (up to 6 years of age).

Many of the patients are either not treated or treated insufficiently because parents do not realize that allergic rhinitis could be an introduction to more serious diseases including asthma. The risk of appearing of symptoms depends on the predisposition towards atopy as well as on the exposure to plant antigens in the pollening season or to mite antigens which "allergize" the all year round. The risk of the disease increases in children born in the months preceding pollen season. The treatment strategy relies on an effective diagnostics and choosing the best mode of treatment in the acute phase of the disease, completing the diagnostics after pollening season and protecting against relapses of the disease.

In the acute phase of the disease the following should be done:

- endoscopy with sample collection to determine eosinophilia in the blood smear,
- rhinomanometric examination to assess the degree of the disease,
- immunological examination to determine the total and possibly specific IgEs,
- skin tests (prick tests with the house dust or food antigens).

The therapy in this stage is based on the elimination of the antigen: removing pollinating plants out of the room, cutting the grass at the patient's place of residence (by non-sensitized persons), closing doors and windows during the pollening period, especially dry and windy one, using pollen absorbers, eliminating the domestic animals' epidermal matter, introducing an elimination diet in case of finding the casual relationship between rhinitis and particular food; in case of allergy to mites - on arranging a dust-free bedroom with the use of appropriate bed linens, removing carpets and dust-accumulating drapes, avoiding excessive humidity and using acaricides. In the pharmacotherapy we use the antihistaminic agents - both parenterally and locally, drugs causing shrinkage of the nasal mucosa (decongestants), corticosteroids and the disodium cromoglycate preparations (Intal) - both for the topical treatment. The antihistaminic agents blocking the H-1 receptor are particularly efficient in removing pruritus, sneezing and excessive nasal secretion, although they do not have a significant influence on nasal congestion. A local administration of these medications is many a time impeded because they are most frequently combined with decongestants - drugs shrinking the mucous membranes with the potential of causing vasocirculatory reaction. That repeatedly makes their use in small children impossible. These medications efficiently eliminate nasal congestion but have little effect on sneezing and rhinitis symptoms. The use of decongestants for more than 14 days can cause rhinitis medicamentosa.

Zyrtec in the form of drops is a very good preparation also for its easiness of precise dosing in small children. In the local therapy, parasympatholytics such as Atrovent, eliminating symptoms connected with the activation of mucous glands, can also be of use. Good effects are achieved through the prolongation of the therapy by using the disodium cromoglycate preparations (Lomusol). This preparation controls rhinorrhea and sneezing by blocking the release of histamine from mast cells and since it lacks any side effects, can be recommended for the treatment during the whole pollination period of the grasses.

At the present a great hope is set on the use of topical corticosteroids. These could be preparations containing beclomethasone dipropionate as an active substance (e.g. Beconase), budesonide, flunisolide, fluticasone propionate (Flixonase) etc. These preparations are most effective in removing nasal congestion. Side effects associated with these medications such as atrophy of the nasal mucosa, sporadic bleedings and tendency to secondary bacterial infections, limit their use in small children. In the period after ceasing the pollination of grasses and weeds, the obtained therapeutic effects should be evaluated in order to select the best preparations which could be used in the following year.

At that time diagnostic investigations could be completed by carrying out skin tests, particularly in case of not being done during the acute phase of the disease, estimation of the total or specific IgE levels. If the analysis of the therapy reveals difficulties in controlling disease manifestations while using specific medications, the introduction of specific immunotherapy in the form of oral preparations in children up to 6 years old, should be considered. The study performed in our institution showed positive effects of this kind of therapy regarding such preparations as Pollinaire mixum (Sevac), Novo Helisen oral (Germany) and Perosall (Biomed, Poland).

A selection of the method of treatment, employing appropriate medications, and determining the limit of their administration time, depends on the physician's experience and on the insight control of the therapy, while positive, lasting effects - on an early introduction of specific desensitization in case of the failure of classic pharmacotherapy.



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THE SPECIFIC IMMUNOTHERAPY IN POLLINOSIS

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The hyposensitisation, now called specific immunotherapy (sIT), was introduced in 1911 by Noon and Freeman for the treatment of patients with pollen rhinitis. Despite such a long tradition sIT remains a very controversial method of therapy, and even now it is not generally accepted. The reasons for this may be found in the lack of scientific bases of sIT, poor knowledge of the underlying mechanisms of both allergic reactions and the action of immunotherapy, and consequently lack of possibility of objective control of the effects of treatment and the frequent occurrence of undesirable symptoms. Without doubt the use of badly purified, nonstandardized allergen extracts played a role in this.

In 1993 a team of experts of EAACI Immunotherapy Subcommittee published the second edition of the Immunotherapy Position Paper, describing the progress in studies of allergic processes and specific immunotherapy mechanisms made in recent years. The Position Paper also gave many practical directions, which can be useful in allergological practice.

According to the majority of allergologists, sIT is still believed to represent an effective and causal treatment of IgE-mediated allergic diseases. The use of sIT in venom and pollen allergy is generally accepted. Pollinosis seems to be a good model for study of the local and systemic allergic process and for evaluating the influence of sIT on this disease.

In considering the direction of sIT action one should take into account: its influence on humoral immunity (IgE, allergen specific IgE and IgG4, anti-IgE), cellular immunity (activity and accumulation of effector cells and release of allerge-inflammatory mediators), and generation of the late allergic reaction phase with participation of eosinophils, neutrophils, basophils and T cells, in particular Th₂. As studies in recent years have shown, sIT influences change in the differentiation of subpopulation Th₂ into Th₁, which is of great importance in the formation of the cytokine release profile and in limiting the two-phase allergic reaction. The Th₂ cells are responsible for production of cytokines such as: IL-3, IL-4, IL-10, which play a significant role in the development of the late allergic reaction, whereas Th₁ cells produce INF- γ and IL-2, which act antagonistically to cytokines produced by Th₂ cells.

At present the characteristic feature of successful sIT is considered to be its ability to inhibit the late allergic reaction induced by the allergen, as has been confirmed in studies of this reaction in the skin, bronchial tree and lately also in the nasal mucosa.

The lecture will also present the clinical results of some studies from the literature and our own experience in sIT in pollinosis, and the future of immunotherapy using recombinant allergens and synthetic peptides, including the major epitopes of given allergens, making safe and effective suppression of specific IgE and inactivation of allergen-specific Th cells possible.



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